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Role of Somatosensory and Vestibular Cues in Attenuating Visually Induced
Human Postural Sway

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Abstract

The purpose of this study was to determine the contribution of visual, vestibular, and somatosensory cues to the maintenance of stance in humans. Postural sway was induced by full field, sinusoidal visual surround rotations about an axis at the level of the ankle joints. The influences of vestibular and somatosensory cues were characterized by comparing postural sway in normal and bilateral vestibular absent subjects in conditions that provided either accurate or inaccurate somatosensory orientation information.

In normal subjects, the amplitude of visually induced sway reached a saturation level as stimulus amplitude increased. The saturation amplitude decreased with increasing stimulus frequency. No saturation phenomena was observed in subjects with vestibular loss, implying that vestibular cues were responsible for the saturation phenomenon. For visually induced sways below the saturation level, the stimulus-response curves for both normal and vestibular loss subjects were nearly identical implying that (1) normal subjects were not using vestibular information to attenuate their visually induced sway, possibly because sway was below a vestibular-related threshold level, and (2) vestibular loss subjects did not utilize visual cues to a greater extent than normal subjects; that is, a fundamental change in visual system "gain" was not used to compensate for a vestibular deficit.

An unexpected finding was that the amplitude of body sway induced by visual surround motion could be almost three times greater than the amplitude of the visual stimulus in normals and vestibular loss subjects. This occurred in conditions where somatosensory cues were inaccurate and at low stimulus amplitudes. A control system model of visually induced postural sway was developed to explain this finding.

For both subject groups, the amplitude of visually induced sway was smaller by a factor of about four in tests where somatosensory cues provided accurate versus inaccurate orientation information. This implied that (1) the vestibular loss subjects did not utilize somatosensory cues to a greater extent than normal subjects; that is, changes in somatosensory system "gain" were not used to compensate for a vestibular deficit, and (2) the threshold for the use of vestibular cues in normals was apparently lower in test conditions where somatosensory cues were providing accurate orientation information.

Key Words: postural control -- vestibular -- somatosensory -- vision -- human

Introduction

Moving visual scenes have long been known to induce postural adjustments in human subjects (Berthoz et al. 1979; Brandt et al. 1986). A wide variety of moving visual stimuli have been employed to investigate this phenomenon including tilting rooms (lateral and fore-aft rotations about an axis at the level of the ankle joints (Bles et al. 1983; Bles et al. 1980)), swinging rooms (Lee and Lishman 1975), projected displays simulating a moving visual wall (van Asten et al. 1988), tunnel, floor, or ceiling (Lestienne et al. 1977; Soechting and Berthoz 1979), and visual roll rotations (Clément et al. 1985; Dichgans et al. 1972).

Direct comparisons among studies are difficult because a variety of visual stimuli have been employed and differing techniques have been used for measuring

body motion. Most experiments have shown that postural adjustments were in the direction of the visual field motion (Berthoz et al. 1979; Bles et al. 1983; Bles et al. 1980; Clément et al. 1985; Dichgans et al. 1972; Lestienne et al. 1977), but oppositely directed body sways have also been reported in some subjects (van Asten et al. 1988). One consistent finding has been the existence of a saturation effect; that is, increases in the amplitude of the visual field movement cause little or no additional postural sway (Bles et al. 1980; Clément et al. 1985; Lestienne et al. 1977; van Asten et al. 1988). Below the saturation level, postural sway deviations have been shown to be proportional to the logarithm of the visual motion amplitude (Lestienne et al. 1977). The stimulus amplitude at which saturation occurs apparently depends upon the availability of accurate somatosensory and vestibular orientation cues. Standing on a compliant surface (foam), which decreases or disrupts somatosensory cues, increases the amplitude of visually induced sway at saturation (Bles et al. 1980). Patients with loss of somatosensation due to polyneuropathy also show increased responsiveness to visual motion (Kotaka et al. 1986). In addition, loss of vestibular function results in increased responsiveness to visual motion stimuli in comparison to normal subjects, although this effect is frequency dependent (Bles et al. 1983).

The purpose of this study was to clarify the role of vestibular and somatosensory information in human postural control by comparing visually induced sway in normal subjects and bilateral vestibular loss patients standing in environments with and without reliable somatosensory cues. This work addressed three specific questions. First, is the increased susceptibility of vestibular loss patients to visually induced sway caused by an increase in visual drive to postural control or due to a loss of attenuation of visually induced sway normally provided by the vestibular system? Second, does a comparison of visually induced sway in normals and vestibular loss subjects reveal the presence of vestibular-related threshold properties in normals? Third, is there evidence for enhanced utilization of somatosensory and/or visual information in order to compensate for a loss of vestibular function?

A portion of these results were previously published in a conference proceedings (Peterka and Benolken 1992).

Materials and methods

The experimental protocols described here were approved by the Institutional Review Board of Legacy Health System and were performed in accordance with the 1964 Helsinki Declaration. All subjects gave their informed consent prior to entering the study. We tested 9 subjects ranging in age from 22 to 67 years. Six subjects (aged 22--45) had normal clinical sensory organization tests of postural control (Peterka and Black 1990) and no history of balance or dizziness complaints. Three subjects (aged 50--67) had bilateral vestibular losses; two were judged to have a profound bilateral loss by the absence of a vestibulo-ocular reflex on rotation tests and severe vestibular dysfunction pattern on sensory organization tests of postural control (Peterka and Black 1990), and 1 had a severe, but not total bilateral loss as judged by a greatly reduced vestibulo-ocular reflex on rotation tests and severe vestibular dysfunction pattern on sensory organization tests. Although the normal and vestibular loss subjects differed in age, control trial measures showed no indication of a difference between the normal and vestibular loss groups in their

utilization of visual and somatosensory cues for balance control. Sensory organization test results for vestibular loss subjects were well within normal limits on test conditions which did not require vestibular cues for balance. Previous work characterizing age-related changes in postural control have identified only minor changes with age in a subject's ability to utilize visual and somatosensory cues (Peterka and Black 1990).

Subjects were tested on a modified Equitest (NeuroCom, Clackamas, OR) moving posture platform. The visual surround on the posture platform rotated under servo control about an axis which was collinear with the ankle joint axis and was located about 10 cm above and 16 cm forward of the ankle joint position. The visual surround was modified to provide a visually provocative scene. The surface of the visual surround facing the subject was located about 65 cm from the subject's eyes and consisted of a circular target pattern of concentric 6.5 cm wide rings of alternating black and white sectors. This pattern was similar to the pattern W2 of van Asten et al. (1988). The right and left sides of the visual surround were located 47 cm from midline and consisted of a checkerboard pattern of alternating black and white rectangles 6.3 by 20.3 cm. Tests were performed in a darkened room with the visual surround illuminated by a fluorescent light attached to the visual surround to keep the illumination level constant as the visual surround moved. Subjects were instructed to maintain upright stance with as little sway as possible. White noise was played into headphones to limit auditory cues to visual surround motion. However, we were not able to fully mask auditory and vibration cues during the largest visual surround motions.

Subject anterior-posterior (AP) sway angle was measured by two horizontal rods attached to the subject at the hip and shoulder level. One end of each rod was attached to the subject and the other end to an earth-fixed potentiometer. The AP displacements of the subject's body at the level of the hips and shoulders were calculated using the output of the two potentiometers with appropriate trigonometric conversions. These measures were used to estimate the body center-of-gravity (CG) displacement using a two part body model which assumed the subject had an average distribution of body mass between the upper and lower body sections (Peterka and Black 1990). The motion of the body was expressed as the angular rotation (in degrees) of the CG point about the ankle joint axis with a positive sign indicating a forward body sway.

Thirty-six tests over three test sessions were performed by each subject. Half of the tests were performed with the subject standing on a fixed support surface, and half on a sway-referenced support surface (see explanation of sway-referencing below). Two tests were control trials performed with eyes open and with no motion of the visual surround. In thirty-four tests the visual surround was sinusoidally rotated at 3 different frequencies (0.1, 0.2 or 0.5 Hz) and 6 different amplitudes (peak displacement amplitudes of 0.2°, 0.5°, 1°, 2°, 5° or 10°). All combinations of frequencies and amplitudes were given except 10° at 0.5 Hz due to motor limitations. The initial motion of the visual surround was always away from the subject. The test sequence was random. An integer number of stimulus cycles was presented over a 60 s duration with a 1 s baseline recorded prior to the start of the stimulus. If a subject fell on a test, that test was immediately repeated up to two more times. Stimulus delivery and data sampling were computer controlled at a clock rate of 50/s. Sampled data included visual surround and support surface

rotational positions, vertical forces exerted on the support surface, and body sway angles at the level of the hip and shoulder.

Sway-referencing was performed by rotating the posture platform's support surface (rotation axis through the ankle joints) in direct proportion to the subject's lower body sway angle as measured by the sway rod attached at the subject's hip. Sway-referencing maintains the ankle joint angle nearly constant over time (assuming no knee flexion). This reduces the contribution of somatosensory cues associated with ankle joint motions which are normally well correlated with body sway when a subject is standing on a fixed surface. The extent to which sway-referencing reduces somatosensory cues is uncertain. However it is certain that the reduction was sufficient to eliminate a vestibular loss subject's ability to maintain upright stance when no other sensory orientation cues were available. This was demonstrated by the consistent falls exhibited by the vestibular loss subjects attempting to stand with eyes closed on a sway-referenced surface. The nature of the falls indicated that no corrective responses were generated, and therefore suggested that somatosensory cues were greatly reduced. In contrast, vestibular loss subjects were able to maintain stance with eyes closed on a fixed support surface. Sway-referencing was initiated at the start of sinusoidal visual surround motion.

The CG sway angle time series was used for the analysis of the steady state responses to the visual stimulus. A Fourier analysis of the steady state CG sway angle and the visual surround angle time series was used to calculate the amplitude of CG sway, amplitude of visual surround motion, and phase of CG sway relative to the stimulus motion. Fourier transforms of the CG sway angle and visual surround angle time series were calculated using the following formulas:

$$\theta_{cg}(f) = \mathcal{F}\{\theta_{cg}(i)\} = \frac{2}{c \cdot N} \sum_{i=1}^{c \cdot N} \theta_{cg}(i) \cos\left(\frac{2\pi fi}{r}\right) - j \frac{2}{c \cdot N} \sum_{i=1}^{c \cdot N} \theta_{cg}(i) \sin\left(\frac{2\pi fi}{r}\right) \quad [1]$$

$$\theta_v(f) = \mathcal{F}\{\theta_v(i)\} \quad [2]$$

where $\theta_{cg}(i)$ is the sampled CG time series, $\theta_v(i)$ is the sampled visual surround angle time series, $j = \sqrt{-1}$, c is the number of stimulus cycles analyzed, N is the number of sample points per stimulus cycle, r is the sampling rate, and f is the frequency (in Hz) at which the Fourier transform was calculated. $\theta_{cg}(f)$ and $\theta_v(f)$ are complex quantities. The amplitude of CG sway and of visual surround motion at a frequency, f , was given by:

$$|\theta_{cg}(f)| = \sqrt{\text{Re}[\theta_{cg}(f)]^2 + \text{Im}[\theta_{cg}(f)]^2} \quad [3]$$

$$|\theta_v(f)| = \sqrt{\text{Re}[\theta_v(f)]^2 + \text{Im}[\theta_v(f)]^2} \quad [4]$$

The transfer function, $H(f)$, between the visual stimulus and the CG body sway was calculated by:

$$H(f) = \frac{\theta_{cg}(f)}{\theta_v(f)} \quad [5]$$

The gain and phase of the transfer function were given by:

$$|H(f)| = \sqrt{\text{Re}[H(f)]^2 + \text{Im}[H(f)]^2} \quad [6]$$

$$\angle H(f) = \tan^{-1} \left(\frac{\text{Im}[H(f)]}{\text{Re}[H(f)]} \right) \quad [7]$$

The first cycle of the CG and visual surround time series were not included in the Fourier analysis in order to avoid transient responses. Since most of this study's results were concerned with the CG sway response to the visual stimulus motion, the Fourier analysis was performed at the frequency of the visual stimulus motion. In some cases $|\theta_{cg}(f)|$ was calculated over a range of frequencies to determine the overall spectrum of CG sway (see Figure 2).

Fourier analysis was also performed on spontaneous CG sway data (eyes open with no visual surround motion) under both fixed and sway-referenced support surface conditions. The spectral amplitudes, $|\theta_{cg}(f)|$, of spontaneous sway at 0.1, 0.2, and 0.5 Hz provided control data for comparison with visually induced sway amplitudes.

Calculations of sway amplitudes and gains using Fourier techniques are potentially biased due to the presence of noise (Bendat and Piersol 1971; Otnes and Enochson 1972). Extensive simulation studies were performed in order to determine if major bias errors were a likely source of error in the calculation of response amplitudes and gains at the stimulus frequency. Using the Fourier analysis methods described above, simulation results showed that bias errors in the measurement of response amplitude were minimal for noise levels that resembled those seen in the experimental data. Bias errors were below 10% of the true response amplitude for noise amplitudes up to 50% of the true response amplitude. Various alternative windowing techniques, cross-spectral analysis methods, and Fourier analysis using segmented data sets were used to determine if biases could be reduced by alternative analysis methods (Otnes and Enochson 1972). None of the alternatives gave lower biases than the analysis used in this study.

Results

The visual surround motion induced a steady state CG sway response at the stimulus frequency and in the same direction as the visual surround motion. Figure 1 shows CG sways induced by the 0.2 Hz, 1° peak amplitude visual surround motion with a sway-referenced support surface. In most traces, a clear response at the stimulus frequency can be seen although response amplitudes varied, particularly among the normal subjects. Figure 2 shows amplitude spectra of CG sway for both normal and vestibular loss subjects during a 0.2 Hz, 1° sinusoidal visual surround rotation. Spectra of CG sway in both sway-referenced (data from Figure 1) and fixed support surface conditions are shown. In addition, amplitude spectra of control trials (eyes open with fixed visual surround) are shown for comparison. A

Fourier analysis (eqns. [1] and [3]) was used to compute the amplitude spectra over a frequency range of 0.05 to 1 Hz.

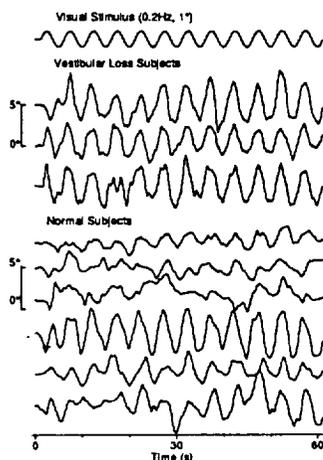


Fig 1. Representative CG sway time series of 3 vestibular loss and 6 normal subjects during 60 s visual surround rotations at 0.2 Hz frequency and 1° peak amplitude with a sway-referenced support surface. Upward deflection represents rotation of the visual surround away from the subject and forward body sway.

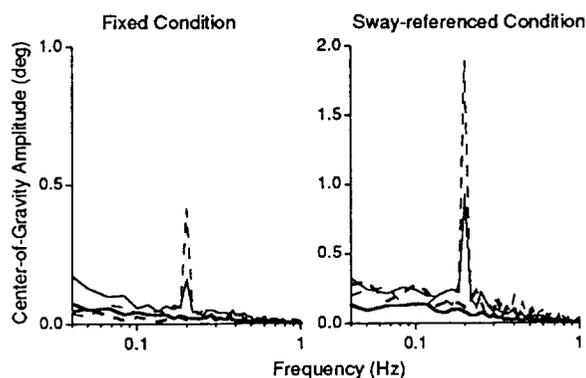


Fig. 2. Example amplitude spectra of CG sway from vestibular loss and normal subjects. The amplitude spectra shown were computed by taking the average of individual test spectra obtained from each subject's CG sway data. Average spectra from the 3 vestibular loss subjects are shown as dashed lines and average spectra from the 6 normals as solid lines. Thick lines indicate spectra from control tests and thin lines indicate spectra from the 0.2 Hz, 1° visual surround motion tests. Amplitude spectra under both fixed support surface conditions (left) and sway-referenced conditions (right) are shown with different scales on the ordinate axes.

The control trial spectra in Figure 2 show that normal and vestibular loss subjects had similar amplitudes and frequency distributions of CG sway. The spectral component of CG sway at the 0.2 Hz visual stimulus frequency was enhanced compared to the control trial amplitude at this frequency. At this specific stimulus frequency and amplitude, the visual surround motion induced about twice the CG sway amplitude in vestibular loss subjects as compared to the normal subjects in the sway-referenced condition, and about 2.5 times the sway in the fixed condition.

In the remainder of the results section, we are concerned only with the Fourier component of sway which is at the stimulus frequency. When we refer to the response amplitude, we are referring to the Fourier component amplitude computed from eqn [3] with f equal to the stimulus frequency.

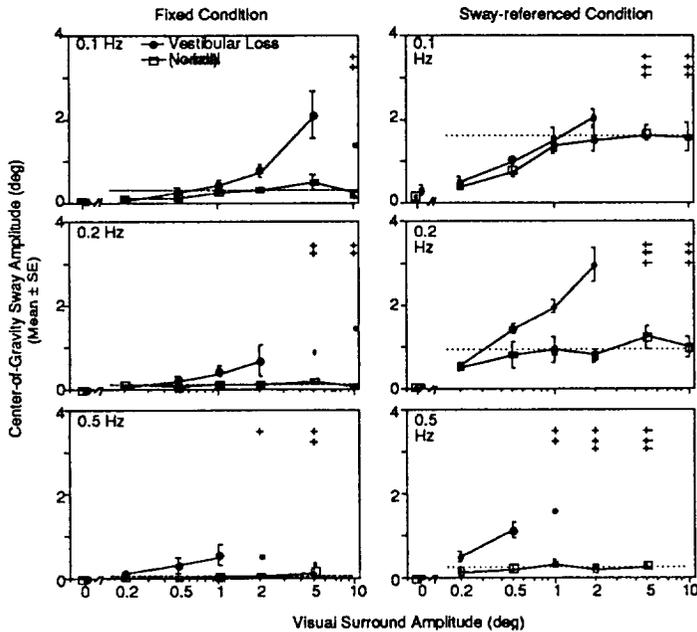


Fig. 3. CG sway amplitude measured at the visual stimulus frequency as a function of visual surround stimulus amplitude under fixed (left column) and sway-referenced (right column) conditions at three frequencies of visual surround motion. The +'s indicate the number of vestibular loss subjects who fell during a given trial. The small dots (7 points) represent the individual data from vestibular loss subjects who did not fall on a particular trial in which others did fall. Six of seven of the individual points, including the 2 lowest amplitude points in the 0.5 Hz fixed condition, are from the vestibular loss subject with the greatest preservation of vestibular function. Data points plotted with error bars (mean \pm 1 standard error) are data from normal subjects (open squares) or data from vestibular loss subjects (large solid dots) where all subjects completed a given trial. Data plotted at zero stimulus amplitude are the control trial spontaneous CG sway amplitudes determined by Fourier analysis at the respective frequencies and support surface conditions. Dashed lines show the average amplitude of saturated sway in the normal subjects.

referenced support surface conditions, with falls occurring at lower stimulus amplitudes when the support surface was sway-referenced. The vestibular loss subject with some preservation of vestibular function (the severe vestibular loss subject) was more resistant to falling than the two subjects with no evidence of vestibular function (profound vestibular losses). The severe vestibular loss subject's resistance to falls was more evident on the fixed support surface trials. Specifically, on 0.1 Hz, 10°, and 0.2 Hz, 5° and 10° fixed support surface trials, his visually induced sway was about six times larger than the average of the normal subjects. However his performance on the 0.5 Hz, 2° and 5° trials was close to the average response of normal subjects. His normal performance at the highest test frequency is consistent with observations that higher frequency vestibular responses are

The amplitude of visually induced sway in normal and vestibular loss subjects was dependent upon stimulus frequency, stimulus amplitude, and the support surface condition. Figure 3 shows the amplitude of CG sway as a function of the visual surround stimulus amplitude for different stimulus frequencies and support surface conditions. In both normal and vestibular loss subjects, visual surround motion induced larger amplitude sways in sway-referenced as compared to fixed support surface conditions at any given stimulus amplitude and frequency.

Normal subjects did not fall on any trial with fixed support surface conditions. Occasional falls occurred among normals during sway-referenced trials, but the normal subjects were always able to complete the trials upon repetition of the test condition. Some of the larger amplitude visual surround motions caused consistent falls in vestibular loss subjects. Vestibular loss subjects fell in both fixed and sway-

preserved relative to lower frequency function in subjects with severe vestibular losses (Honrubia et al. 1985).

Saturation and threshold phenomena

Among normal subjects tested under sway-referenced conditions (Figure 3, right column), a saturation in the amplitude of visually induced CG sway occurred as the visual stimulus amplitude increased (most evident in the 0.1 Hz sway-referenced data). The saturation level decreased with increasing stimulus frequency. Below the saturation level, CG sway increased in proportion to the logarithm of the visual stimulus amplitude. For the vestibular loss subjects there was no saturation effect. That is, visually induced sway increased as a function of the logarithm of the stimulus amplitude until falls occurred at higher stimulus amplitudes.

For sway-referenced test conditions which evoked responses with amplitudes below the saturation levels, the amplitude of visually induced sway was similar in normal and vestibular loss subjects up to the point where the normal subjects reached the saturation level. Specifically, the 0.1 Hz sway-referenced data showed similar amplitudes of induced sways in normals and vestibular loss subjects for stimulus amplitudes of 0.2°, 0.5°, and 1°, but a clear divergence between normals and vestibular loss subjects at stimulus amplitudes of 2° and above (falls occurred in vestibular loss subjects with 5° and 10° stimuli). At 0.2 Hz, the induced sways were nearly identical for normals and vestibular loss subjects only at the lowest stimulus amplitude of 0.2°, with a clear divergence between the test groups at higher stimulus amplitudes. At 0.5 Hz, visually induced sway in vestibular loss subjects was greater than in normal subjects even at the lowest stimulus amplitude of 0.2°.

Visually induced sway under fixed support surface conditions (Figure 3, left column) showed a similar pattern of frequency and amplitude dependence as in the sway-referenced condition. However, under fixed support surface conditions the amplitudes of visually induced sway were about four times lower than in corresponding sway-referenced conditions. As in the sway-referenced condition, there was a saturation effect in the normal group at higher stimulus amplitudes. For stimuli above the saturation level at any given frequency, there was a clear divergence between the normal and vestibular loss groups in the amplitude of visually induced sway. The vestibular loss group showed increasing sway with increasing stimulus amplitude (with falls occurring at higher stimulus amplitudes). Below the saturation levels, the induced sways were similar in the two groups.

This saturation phenomenon and the correspondence between the sway amplitudes in normal and vestibular loss subjects at low stimulus amplitudes suggests that normal subjects were not making use of vestibular motion cues at these low sway amplitudes. That is, at sways below a threshold amplitude, normal subjects did not attenuate visually induced sway more than vestibular loss subjects. These vestibular-related threshold amplitudes were estimated by averaging the CG sway amplitudes at high stimulus amplitudes where the sway versus stimulus amplitude curves for normal subjects diverged from those of the vestibular loss subjects and saturated. The choice of which stimulus amplitude points to include in these averages was based on visual inspection of the various curves. In each of the fixed condition tests the data from the highest 4 stimulus amplitudes were included in the average. In the sway-referenced condition tests the 3, 5, and 4 highest

stimulus amplitude data at 0.1, 0.2, and 0.5 Hz, respectively, were included in the average. These threshold amplitudes are given in Table 1 and plotted as dotted lines in Figure 3. The threshold amplitudes decreased with increasing stimulus frequency in both fixed and sway-referenced conditions. Under fixed support surface conditions, the threshold estimates were 3--5 times lower than under sway-referenced support surface conditions.

Table 1. Threshold estimates (saturated CG sway amplitude) in normal subjects (mean \pm SE)

Frequency (Hz)	Fixed Condition		Sway-referenced Condition	
	Angular position (deg)	Angular velocity ($^{\circ}$ /s)	Angular position (deg)	Angular velocity ($^{\circ}$ /s)
0.1	0.32 \pm 0.06	0.20 \pm 0.04	1.57 \pm 0.15	0.99 \pm 0.09
0.2	0.17 \pm 0.02	0.21 \pm 0.03	0.98 \pm 0.11	1.23 \pm 0.14
0.5	0.10 \pm 0.02	0.31 \pm 0.06	0.29 \pm 0.04	0.92 \pm 0.11

Response gain and phase.

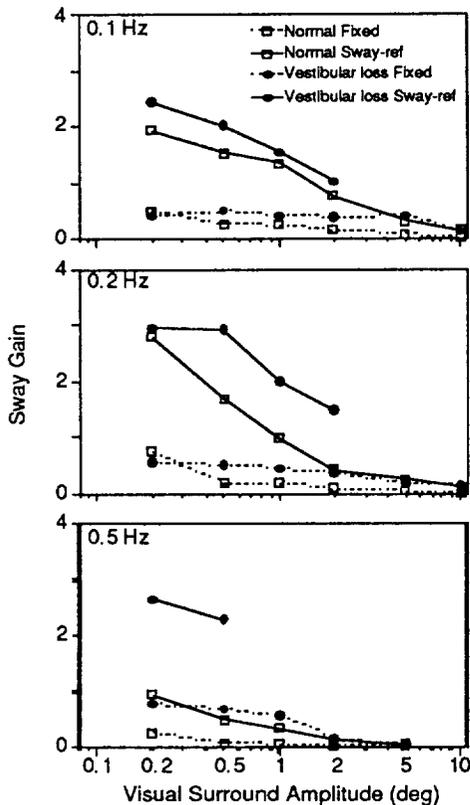


Fig. 4. Mean gain (ratio of CG sway amplitude at the stimulus frequency to visual surround stimulus amplitude) of visually induced CG sway as a function of visual surround amplitude at three frequencies of visual surround motion.

Visually induced postural responses can also be expressed in terms of response gain and phase (eqns [6] and [7]). In fixed support surface test conditions, the gains for both normal and vestibular loss subjects were less than unity at all stimulus amplitudes and frequencies tested (Figure 4). At a given stimulus frequency, gains of both normal and vestibular loss subjects generally decreased with increasing stimulus amplitude. Because of the saturation phenomenon seen in normal subjects, the gains of normals decreased more rapidly with increasing stimulus amplitude than did the gains of vestibular loss subjects.

In sway-referenced support surface test conditions, the response gains of vestibular loss subjects were greater than unity at all test frequencies and amplitudes where data were obtained. The gains were greatest at the lowest test amplitude of 0.2 $^{\circ}$ where the average gains were 2.4, 2.9, and 2.6 at frequencies 0.1, 0.2, and 0.5 Hz, respectively. Normal subjects also had gains greater than or equal to unity at the lowest test amplitude of 0.2 $^{\circ}$ (average gains of 1.9, 2.8, and 1.0 at frequencies 0.1, 0.2, and 0.5 Hz, respectively). The average gains of normals were less than unity at stimulus amplitudes greater than or equal to 2 $^{\circ}$, 1 $^{\circ}$, and 0.5 $^{\circ}$ at 0.1, 0.2, and 0.5 Hz, respectively.

The phase of the CG sway angle relative to the stimulus angular position is summarized in Table 2. Phase data were averaged over all completed trials of a given test frequency and condition. The data show two general trends. First, phases for both normal and vestibular loss subjects decreased

(increased phase lag) with increasing stimulus frequency. Second, at any given frequency and support surface condition, the average phase of vestibular loss subjects was advanced relative to the average phase of normal subjects.

Table 2. Phase of CG sway relative to visual surround position (degrees, mean \pm SE)

Frequency (Hz)	Fixed Condition		Sway-referenced Condition	
	Normal	Vestibular Loss	Normal	Vestibular Loss
0.1	-12 \pm 16	21 \pm 6	19 \pm 11	41 \pm 5
0.2	-28 \pm 23	0 \pm 6	-30 \pm 13	11 \pm 11
0.5	-37 \pm 32	-16 \pm 17	-87 \pm 17	-81 \pm 15

The averaging of phase over all stimulus amplitude trials was justified at most test frequencies and conditions since phase did not vary systematically with stimulus amplitude. However there were exceptions. Specifically, the phases of normal subjects in 0.1 Hz and 0.2 Hz sway-referenced conditions and in 0.1 Hz fixed conditions showed significant trends ($P < 0.05$) of decreasing phase with increasing stimulus amplitude. The trends were approximately proportional to the logarithm of the stimulus amplitude. In all three of these data sets, the phase of the normal subjects at the 0.2° test amplitude was closest to, but lagged the vestibular loss subjects' phase. At higher stimulus amplitudes, the normal and vestibular loss subjects' phases diverged since the vestibular loss subjects' phase remained constant while the normal subjects' phase decreased.

The only test condition which showed a significant phase trend in the vestibular loss subjects was the 0.5 Hz sway-referenced condition. This trend was for increasing phase with increasing stimulus amplitude. However this data set was limited to only the lowest stimulus amplitudes since falls consistently occurred at the higher amplitudes.

Dependence upon somatosensory cues

A comparison of subject performance under fixed versus sway-referenced conditions provided information on the extent to which the availability of accurate somatosensory cues decreased visually induced sway. This comparison was quantified by computing the ratio of visually induced sway amplitude under fixed support surface conditions to the sway amplitude under sway-referenced conditions in normal and vestibular loss subjects. The ratio was computed using only the amplitude component of sway at the stimulus frequency. This ratio did not show any trend with stimulus frequency or amplitude, and was nearly identical for the normal and vestibular loss groups. The average ratio for normals over all test conditions was 0.24 ± 0.18 (mean \pm 1 SD) and for vestibular loss subjects was 0.24 ± 0.14 . That is, a decrease in the accuracy of somatosensory cues caused a degradation of postural stability by the same factor for both normal and vestibular loss subjects. This suggested that the vestibular loss subjects had not become more reliant upon somatosensory cues (i.e. had not increased somatosensory gain) to compensate for their vestibular loss.

Discussion

High gains of visually induced sway

The existence of gains greater than unity for visually induced sway was an unexpected finding. These high gain responses occurred in both normal and vestibular loss subjects under conditions where somatosensory orientation cues were inaccurate (sway-referenced support surface conditions) and at low amplitudes of visual surround motion. We are not aware of any description of high gains of visually induced sway in previous studies. This is likely due to the fact that most studies used larger amplitude visual surround motions which evoked proportionally lower amplitudes of body sway (this is consistent with our data at higher stimulus amplitudes). In addition, many studies used center-of-pressure energy measures which do not permit a calculation of sway gains. However one study (Lee and Lishman 1975) showed an example figure of sway velocity and visual surround stimulus velocity for a subject standing on a thick foam pad and exposed to a low amplitude, 0.25 Hz sinusoidal AP oscillations of a visual surround. Although the stimulus was a linear translational visual surround movement, for comparison purposes this movement would correspond to about a 0.1° visual surround tilt amplitude about the ankle joints. The induced sway velocity was clearly greater than the stimulus velocity with an estimated gain of about 1.5.

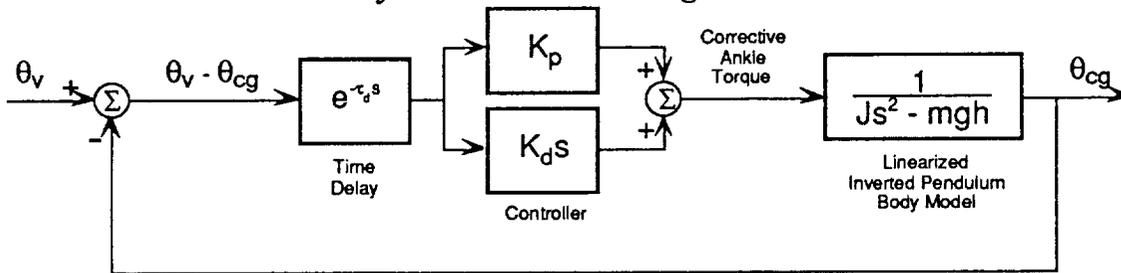


Fig. 5. Simple linearized postural control system model of CG body sway, θ_{cg} , induced by visual surround motion, θ_v . The model assumes that somatosensory and vestibular cues are not contributing to body stabilization. Various model parameters are defined in the discussion section (eqn [8]).

Consideration of the simplest possible model of postural control gives some validity to the existence of high gains during visual motion stimulation. The control system model in Figure 5 models the human body as an inverted pendulum. The model assumes there is no contribution of either somatosensory or vestibular motion cues to the feedback control of the inverted pendulum. The body sway angle relative to the visual surround angle ($\theta_v - \theta_{cg}$) is detected by the visual system with a time delay of 0.2 seconds (visual processing and transmission delays). A corrective torque about the ankle joint must be applied to maintain stability, and this corrective torque is a function of $\theta_v - \theta_{cg}$. In order for this system to be stable, it is known (Johansson et al. 1988; Johansson, 1993) that the function of $\theta_v - \theta_{cg}$ must include at least two terms; one proportional to $\theta_v - \theta_{cg}$ and one proportional to the time derivative of $\theta_v - \theta_{cg}$. With these two terms present, the overall transfer function of this model is given by:

$$\frac{\theta_{cg}(s)}{\theta_v(s)} = \frac{(K_d s + K_p) e^{-\tau_d s}}{J s^2 + (K_d s + K_p) e^{-\tau_d s} - mgh} \quad [8]$$

where s is the Laplace transform variable, J is the moment of inertia of the body about the ankle joint, m is the mass of the body, h is the height of the body's center-of-gravity, g is acceleration due to gravity, τ_d is a time delay, K_p is the proportionality constant of $\theta_v - \theta_{cg}$, and K_d is the proportionality constant of the time derivative of $\theta_v - \theta_{cg}$. J and mgh values of 62 kg-m^2 and $540 \text{ kg-m}^2/\text{s}^2$, respectively, are reasonable estimates for subjects in this study (average mass 64 kg). The predicted DC gain of this system (at $s = 0$) is equal to $K_p/(K_p - mgh)$ and is therefore greater than unity. Furthermore, only a limited range of K_p and K_d values are consistent with a stable system. The values K_p and K_d at the midpoint of their stable ranges are 830 N-m/rad and 345 N-m-s/rad , respectively. With these values, the model predicts a DC gain of 2.9. In addition, the transfer function gain is nearly constant at up to a frequency of 0.4 Hz. Above 0.4 Hz, the gain rises slowly to a weak resonant peak at about 0.8 Hz and then declines.

The predicted transfer function gain values are reasonably consistent with the gains observed in vestibular loss subjects at low stimulus amplitudes in the sway-referenced condition at all test frequencies. The predicted gains are also similar to the low stimulus amplitude gains of normals at 0.1 and 0.2 Hz in the sway-referenced condition, but not at 0.5 Hz. This discrepancy at 0.5 Hz suggests that the simple model in Figure 5, which assumes no somatosensory or vestibular feedback, does not accurately represent the system at this test frequency. There is no reason to expect that the somatosensory cues differ between the vestibular loss and normal subjects at 0.5 Hz. However if a vestibular-related threshold phenomena were present and the threshold level decreased with increasing frequency, then vestibular motion information could contribute to postural stabilization in normal subjects during a 0.5 Hz low amplitude stimulus, but not during a 0.1 and 0.2 Hz low amplitude stimulus (see further discussion below).

Saturation and attenuation phenomena

CG sways of normal subjects induced by visual surround motion showed nonlinear stimulus-response relations and saturation phenomenon such that increasing amplitudes of visual surround motion did not evoke increasing CG sway. In subjects with bilaterally absent vestibular function, this saturation phenomenon was completely absent. In these vestibular loss subjects, increasing visual surround motion induced increasing CG sways, resulting in consistent falls at larger stimulus amplitudes even when accurate somatosensory orientation cues were present (fixed support surface conditions). This implies that the sensory cues which cause this saturation phenomenon are of vestibular origin.

The vestibular contribution to the attenuation of visually induced sway was very different from the somatosensory contribution. For both normal and vestibular loss subjects, the availability of accurate somatosensory cues (fixed support surface conditions) resulted in a fourfold decrease in visually induced sway gains in comparison to test conditions with inaccurate somatosensory cues (sway-referenced conditions). This somatosensory-related attenuation of gain was independent of the

stimulus amplitude and frequency, and occurred in both normal and vestibular loss subjects. In contrast, the saturation phenomenon associated with the availability of vestibular cues showed specific changes as a function of the stimulus frequency and amplitude (the saturation sway amplitude decreased with increasing frequency).

A saturation phenomenon has different functional consequences than a simple gain attenuation. If a subject is exposed to an environment with inaccurate visual orientation cues, the availability of an additional sensory orientation cue which decreases visually induced sway gain can decrease the likelihood of a fall, but cannot prevent a loss of balance. That is, a large amplitude visual motion stimulus can always overcome the decreased gain. In contrast, a saturation effect can completely prevent a loss of balance independent of the visual stimulus amplitude as long as the CG sway amplitude at saturation is within the normal stance range.

Threshold phenomenon

A comparison of the visually induced sway amplitude in normal and vestibular loss subjects showed that the induced sway in normals and vestibular loss subjects was similar until some critical, or threshold level of CG sway was reached. This implies that normal subjects were not making use of vestibular motion information to attenuate visually induced sway until some threshold CG sway amplitude was exceeded.

Since head motions were not measured in these experiments, the observed threshold phenomenon cannot be directly attributed to the vestibular system alone. Studies have shown that head orientation in space tends to be stabilized during various locomotor tasks (Grossman et al. 1988; Pozzo et al. 1990). Therefore the angular or linear head motion components sensed by the vestibular system could be reduced and altered compared to head motions predicted from CG sway measures which assume that the head is rigidly fixed to the body. If there were changes in head position with respect to the trunk during sway, then the observed threshold effects might be related to an interplay between proprioceptive head motion information from cervical afferents and vestibular motion information.

Mergner and coworkers (1991) hypothesized that, in some cases, information from vestibular and neck proprioceptive systems used for motion perception is combined and processed through the same central thresholding neural circuitry as is vestibular motion information alone. If the postural control system uses similar mechanisms for processing combined vestibular and proprioceptive information, then it is possible that the threshold properties derived from our visually induced sway measures might be similar to vestibular thresholds identified by others using psychophysical measures.

In both fixed and sway-referenced support surface conditions, peak angular position threshold amplitudes declined with increasing frequency. When these threshold measures were expressed in terms of angular velocity, their values were approximately equal at the three test frequencies for a given support surface condition (average amplitude of 1.05°/s or 2.1°/s peak-to-peak in the sway-referenced condition, Table 1). This value is close to psychophysically derived rotational motion thresholds (Benson et al. 1989; Benson and Brown 1992; Mergner et al. 1991) which, when expressed in terms of angular velocity, are also relatively constant over the range of test frequencies used in this study (0.1--0.5 Hz).

Under fixed support surface conditions, there was an apparent fourfold reduction in threshold amplitudes (average amplitude of 0.24°/s or 0.48°/s peak-to-peak in the fixed condition, Table 1) relative to the sway-referenced condition. This fourfold reduction could be analogous to the observation that angular motion perception thresholds were lower by a factor of about three in experimental situations which evoked the oculogyral illusion in comparison to perceptual thresholds measured in complete darkness (Benson and Brown 1989, 1992; Clark and Stewart 1968). Another possible analogous situation, identified by Mergner and coworkers (1991, 1993), is a task dependent change in threshold amplitudes associated with the processing of neck and leg proprioceptive information for the perception of relative movement of various body segments.

What is the cause of the fourfold threshold shift associated with different support surface conditions? One could speculate that the central postural system actively adjusts thresholds to optimize balance control under varying environmental conditions. For example, when visual and somatosensory orientation cues are absent or inaccurate, it may be advantageous to have increased thresholds in order to avoid vestibular-initiated control actions caused by small imbalances or asymmetries of peripheral vestibular function. When other accurate sensory orientation cues are present, these other cues might serve as a reference for vestibular signals so that vestibular-initiated control actions could occur at lower levels of body sway. Alternatively, the apparent vestibular threshold shifts might be due to changes in the signal-to-noise ratio of vestibular signals. For example, head movements associated with spontaneous body sway and due to the inherent instability of the head-neck system (Goldberg 1992) would generate a baseline level of "vestibular noise". In situations where accurate sensory orientation cues were available from the visual and somatosensory systems, head stability in space would be improved and therefore vestibular noise reduced. If postural responses were evoked only when a vestibular signal rose above the baseline noise level, then postural responses due to vestibular stimulation would be observed at lower stimulus levels in low vestibular noise conditions (i.e. when accurate visual and somatosensory cues were present) compared to high vestibular noise conditions.

Other evidence of threshold effects in postural control have recently been observed. Collins and De Luca (1993) analyzed center-of-pressure time series data recorded during quiet stance. Their results suggested that short term postural fluctuations were not controlled by closed-loop mechanisms until some systematic threshold was exceeded. The existence of a threshold for the use of vestibular information might contribute to the open-loop/closed-loop control strategy identified by Collins and De Luca.

Compensation for Vestibular Loss

One might expect that well-compensated vestibular loss subjects would adjust to their vestibular loss by altering the way in which somatosensory and/or visual cues are used for postural control. That is, the appropriate strategy for using somatosensory and visual sensory information for balance control might be different when vestibular cues are not available, and central mechanisms might adapt to achieve a more optimal utilization of the available visual and somatosensory sensory cues.

Bles et al. (1983) tracked visually induced lateral body tilts over time in one patient following a bilateral loss of vestibular function. The results generally showed decreases in the amplitude of induced sway over time which suggested that somatosensory cues were becoming more effective in attenuating the visually induced sway. However the attenuation was frequency dependent with the greatest attenuation changes over time occurring at the lowest test frequency (0.025 Hz), and no attenuation occurring at the highest (0.2 Hz). Our test frequencies corresponded to the upper range of frequencies used by Bles et al. (1983). At these higher frequencies we also were not able to identify compensatory changes in the use of somatosensory cues in vestibular loss subjects.

If vestibular loss subjects compensated by increasing their sensitivity to somatosensory cues for balance control, then the loss of somatosensory cues or a decrease in accuracy of those cues should have a proportionally larger effect on their balance than it does on normal subjects. Our data showed that this was not the case since both normal and vestibular loss subjects had nearly identical factor of four reductions of sway in the fixed versus the sway-referenced condition. This suggests that the vestibular loss subjects have not experienced a change in sensitivity to somatosensory cues as a result of their loss of vestibular function over the frequency range tested. As mentioned above, Bles et al. (1983) identified compensatory changes at lower test frequencies and in more dynamic settings (Bles et al. 1984).

If one were to compare the sway amplitudes in normal and vestibular loss subjects during larger amplitude visual motion stimuli, one might conclude that visual motion sensitivity was increased in vestibular loss subjects. This increased sensitivity might be the result of central compensatory adjustments for the loss of vestibular function. However the data in Figure 3 indicate that low amplitude visual surround movements induced approximately equal CG sway amplitudes in both normals and vestibular loss subjects. This close correspondence between the levels of induced sway in normal and vestibular loss subjects at low stimulus amplitudes suggests that the apparent increase in visual sensitivity in vestibular loss subjects is likely due to the absence of vestibular suppression of postural sway rather than to a fundamental increase in sensitivity to visual motion.

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Simple Models of Sensory Interaction in Human Postural Control

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INTRODUCTION

Upright body stance is inherently unstable. Active feedback control utilizing motion cues from various sensory systems is necessary in order to maintain this upright position. Visual, somatosensory, and vestibular sensory systems are the main contributors to this feedback control. In many environments, accurate information is available from all three of these sensory systems. In other environments, motion information from one or more sensory systems may be absent or inaccurate leading to poor performance (increased body sway) or falls in extreme cases. However simple observations indicate that the motion information from the three sensory systems is highly redundant in that upright stance can be maintained when orientation cues are absent or inaccurate in two of the three sensory systems. For example, a normal subject can stand with eyes closed on a foam pad suggesting that vestibular cues alone are sufficient to maintain upright stance. Also, a subject with a bilateral vestibular loss can stand on a flat surface with eyes closed indicating that somatosensory cues alone provide sufficient feedback information for postural control.

Although the influences of sensory cues on postural sway have been clearly demonstrated (Lestienne et al., 1977; Diener et al., 1984; Dichgans & Diener, 1989), very little is known about how visual, somatosensory, and vestibular motion information is combined and processed by the nervous system for postural control. There are many relevant questions. (1) How does the postural control system deal with conflicting sensory orientation information? The postural control system might be capable of assessing the accuracy of available sensory cues and excluding information which is judged to be inaccurate. Alternatively, information from all three sensory systems might be used continuously, but with postural control system parameters set to minimize postural disturbances due to inaccurate sensory information. (2) Are the sensory system interactions linear in nature? Mergner and coworkers (Mergner et al., 1991) have demonstrated that the perception of combined head and body movements can be explained by an essentially linear interaction of vestibular and somatosensory motion cues. Perhaps the postural control system makes use of the same or similar neural mechanisms for combining motion cues. (3) What types of sensory information processing are necessary for postural control?

The purpose of the present work was to develop a control system model-based approach to the study of sensory interactions in postural control. The models serve as an explicit hypothesis regarding the functional mechanisms involved in the processing of information for postural control. An important goal of the modeling work is to develop an intuitive understanding of how the entire system functions and how individual components influence the overall system's responses.

MODELING ASSUMPTIONS

There are many complications in the application of control systems modeling to the postural control problem. First, it is known that various components of the postural system are nonlinear. Nonlinearities can arise from several sources including body mechanics, sensory system response properties, central processing of sensory signals, time delays in neural processing and transmission, and muscle activation properties. Second, if the body is accurately modeled as a multi-link structure, the equations of motion become very complex (Koozekanani et al., 1980). This inherent complexity can defeat the goal of obtaining intuitive insight into the functioning of the postural control system. Third, limited data are available for the validation of any model.

To overcome these problems, our first attempt at modeling and experimental validation of a model was to simplify the system as much as possible. This was accomplished in several ways. First, the vestibular contribution to postural control was not included. Experimental results for model validation came from subjects with a bilateral loss of vestibular function. Second, the body was modeled as an inverted pendulum (single body segment) with rotational motions occurring about the ankle joints in only one plane (anterior-posterior body sway). An inverted pendulum is described by a nonlinear equation of motion. This equation was linearized assuming small angles of rotation about the ankle joint (Ishida & Miyazaki, 1987). Third, the model only attempted to describe the small amplitude responses of the system. Many nonlinear systems effectively behave in a linear manner for small amplitude perturbations. (Exceptions include systems with significant dead-zone and threshold properties.) Fourth, the somatosensory and visual information used for postural control were assumed to be processed independently of one another and then combined linearly. Fifth, the central processing of sensory "error" signals were assumed to be by a simple controller mechanism which generates corrective body torque in proportion to a linear combination of position error, velocity error, and the integral of position error. This type of control processing is known as a PID controller (proportional, integral, differential control) and has been used previously to model human postural control properties (Johansson et al., 1988).

MODEL DESCRIPTION

The block diagram model for the postural control system is shown in Figure 1. It consists of two feedback loops, one associated with the processing of somatosensory orientation cues, and one associated with visual orientation cues. There are two inputs to the model. One is the rotation angle, q_p , of the platform (support surface) upon which the subject stands. The second is the rotation angle, θ_v , of a visual surround which encloses the test subject. The rotation axes of the visual surround and the support surface are assumed to pass through the subject's ankle joint axis.

The subject's body is assumed to be an inverted pendulum, that is, a rigid structure which rotates only in an anterior-posterior direction about the ankle joint axis. The differential equation describing this system is:

$$\begin{aligned} J \frac{d^2\theta_b}{dt^2} &= mgh \sin\theta_b - T \\ &\approx mgh\theta_b - T \end{aligned} \quad [1]$$

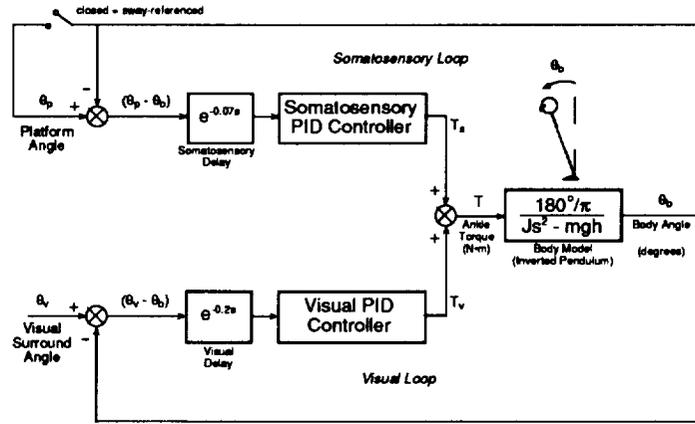


Figure 1. Control system model of human postural control which includes only somatosensory and visual feedback.

where θ_b is the angular deviation of the body away from vertical, J is the body's moment of inertia about the ankle joint, m is body mass, h is the height of the center-of-gravity above the ankle joint, g is the acceleration due to gravity, and T is torque exerted about the ankle joint to maintain stability. The equation is nonlinear due to the $\sin\theta_b$ term, but it can be easily linearized by approximating $\sin\theta_b$ with θ_b for small values of θ_b . If no corrective torque is applied ($T = 0$), the inverted pendulum model is inherently unstable since any small deviation of the body from upright, for example in a positive direction, results in a positive acceleration of the body in the same positive direction. The transfer function equation of the linearized differential equation is given by:

$$\frac{\theta_b(s)}{-T(s)} = \frac{1}{Js^2 - mgh} \quad [2]$$

where s is the Laplace transform variable.

The corrective ankle torque, T , is generated in proportion to "error" signals associated with changes in θ_b . The error signal from somatosensory cues is proportional to the difference between the support surface position and the body sway angle ($\theta_p - \theta_b$). Similarly, the error signal from visual cues is proportional to ($\theta_v - \theta_b$). The error signals are processed through time delay elements representing neural processing, transmission, and muscle activation delays. In addition the error signals are transformed by PID controllers and used to generate corrective torques about the ankle joint. The equation representing PID function for the visual feedback loop is given by:

$$T_v(t) = K_{pv}(\theta_v - \theta_b) + K_{iv} \int_0^t (\theta_v - \theta_b) dt + K_{dv} \frac{d(\theta_v - \theta_b)}{dt} \quad [3]$$

where T_v is the corrective torque generated by the visual feedback loop, and K_{pv} , K_{iv} , and K_{dv} are constants. Similarly, the PID controller generates the corrective torque T_s with PID controller constants K_{ps} , K_{is} , and K_{ds} . The two corrective torques are summed to produce the total corrective torque acting on the body to maintain stability.

Finally, a switch is shown in the somatosensory feedback loop. This switch represents an experimental test condition referred to as "sway-referencing". When this switch is closed, the body sway angle measure, θ_b , is used to drive a servo-control system that rotates the support surface in direct proportion to θ_b . This effectively main-

tains the somatosensory error signal ($\theta_p - \theta_b$) at a very low value. If, for modeling purposes, we assume that ($\theta_p - \theta_b$) is zero during sway-referencing, then T_s is also zero, and the somatosensory loop is no longer contributing corrective torque for postural control. Therefore the model assumes that only visual motions cues contribute to balance control in the sway-referenced test condition for vestibular loss subjects.

MODEL TUNING AND PREDICTIONS

The dynamic properties of the model are easily summarized by computing the model's overall transfer function. The simplest possible example is for the sway-referenced condition. In addition, assume that the visual PID controller only has proportional and derivative components (i.e. $K_{iv} = 0$). This assumption is initially made because it is known that only proportional and derivative components are necessary for the stable control of an inverted pendulum (Johansson et al., 1988). The transfer function equation for this system is given by:

$$\frac{\theta_b(s)}{\theta_v(s)} = \frac{(K_{dv}s + K_{pv})e^{-\tau_{dv}s}}{Js^2 + K_{dv}e^{-\tau_{dv}s}s + K_{pv}e^{-\tau_{dv}s} - mgh} \quad [4]$$

where τ_{dv} is the time delay associated with visual motion processing. This transfer function can be used to predict the amplitude and timing (phase) of θ_b in response to a sinusoidal visual surround motion stimulus at varying stimulus frequencies. The gain is defined as the response amplitude divided by stimulus amplitude. The DC gain (obtained by setting $s=0$ in the transfer function equation) of this transfer function is equal to $K_{pv} / (K_{pv} - mgh)$ and is therefore greater than unity. That is, the model predicts that DC or very low frequency sinusoidal tilting motions of the visual surround will evoke body sways that are greater than the visual surround motion itself!

If the integral component is added back into the PID controller, then the overall transfer function is given by:

$$\frac{\theta_b(s)}{\theta_v(s)} = \frac{(K_{dv}s^2 + K_{pv}s + K_{iv})e^{-\tau_{dv}s}}{Js^3 + K_{dv}e^{-\tau_{dv}s}s^2 + (K_{pv}e^{-\tau_{dv}s} - mgh)s + K_{iv}e^{-\tau_{dv}s}} \quad [5]$$

This transfer function has unity gain at DC since the function of integral control action is to reduce steady state error ($\theta_v - \theta_b$) to zero. However at higher stimulus frequencies, the gain of the transfer function can still be greater than unity. This can be seen in Figure 2 which shows a family of curves, each one graphing gain as a function of stimulus frequency. In all of these curves, the gains remain above unity except for frequencies above about 1.5 Hz. The shape of the various gain curves depends on the values of the PID control parameters. Furthermore, the range of PID parameter values consistent with stable operation of the overall control system is limited. The gain curves in Figure 2 were calculated by keeping visual controller parameters K_{dv} and K_{iv} at fixed values while varying K_{pv} over its entire range of stable operation. As K_{pv} decreases, a sharp resonant peak at about 0.2 Hz develops. The overall system becomes unstable for values of K_{pv} less than about 11 N-m/deg. As K_{pv} increases, a sharp resonant peak at about 0.8 Hz develops, and the system is unstable if K_{pv} is greater than about 19 N-m/deg. A reasonable prediction is that the nervous system would select controller parameters which are near the middle of the stable range, and would therefore avoid the strong resonance created when a parameter is near a stability limit.

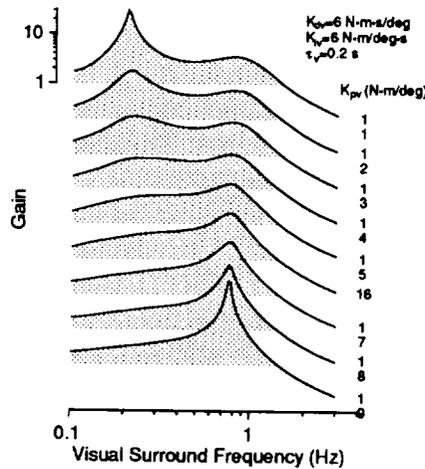


Figure 2. Predicted transfer function gain (body sway amplitude divided by visual surround amplitude) as a function of visual surround stimulus frequency for tests performed under sway-referenced conditions. The family of curves are calculated from Equation 5. The curves show the results of varying the visual PID controller parameter, K_{pv} , over a range consistent with stable operation while other PID parameters remain fixed. The gain scale applies to the upper gain curve and other curves are offset from one another by a gain factor of 5. The shaded areas indicate portions of the curves where gains are greater than unity.

If the posture platform support surface is kept in a fixed position (open switch in Figure 1 with $\theta_p = 0$), then $(\theta_p - \theta_b)$ changes with body sway and the somatosensory controller generates torque, T_s , which contributes to postural control in addition to T_v . A transfer function equation relating body sway to visual surround motion can be written for the entire system. This transfer function equation is more complicated than the one for the visual loop alone. The transfer function gain curves for the combined somatosensory-visual loop system show similar properties to those in Figure 2 in that resonant peaks emerge as somatosensory control parameters approach their stability limits. However, in contrast to the visual loop transfer functions in which gains are generally greater than unity, the gains of the combined somatosensory-visual loop system are generally less than unity.

COMPARISON WITH EXPERIMENTAL DATA

One might anticipate that all of the assumptions, simplifications, and approximations used in the modeling of postural responses would leave little chance that the modeling results could correspond with actual experimental observations. However this is not the case. Figure 3 shows gain and phase data at 0.1, 0.2, and 0.5 Hz obtained from 3 subjects with bilateral vestibular losses. The gain and phase data were calculated from center-of-gravity body sway angle responses to visual surround rotations at 0.1, 0.2, and 0.5 Hz. The data are only from responses to low amplitude visual motion stimuli ($\pm 0.2^\circ$ and $\pm 0.5^\circ$) in order to avoid nonlinear responses associated with larger stimuli. The data in the right column of Figure 3 are from tests performed with a sway-referenced support surface, and in the left column with a fixed support surface. For the sway-referenced condition, experimental data have gain values between 2 and 3, and there is a phase lead of body sway relative to visual surround position at 0.1 and 0.2 Hz. With the visual time delay parameter set to 0.2 s, the visual loop PID parameters in Equation 5 can be found which provide a reasonable fit to the available gain

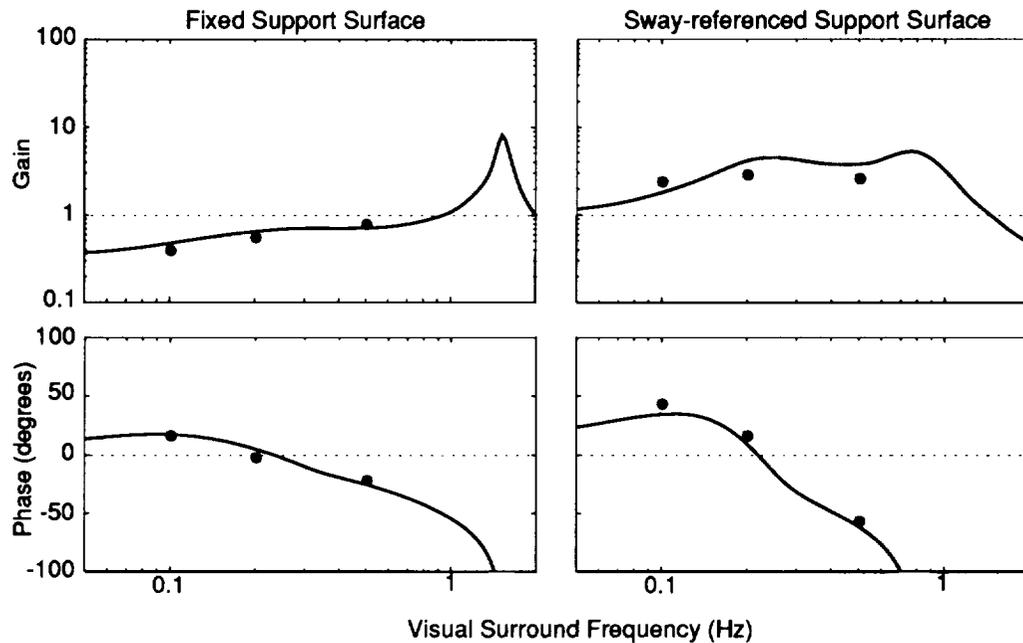


Figure 3. Gain and phase of body sway evoked by visual surround motions of varying frequencies and in conditions with a fixed (left column) or sway-referenced (right column) support surface. Solid dots represent average data from 3 subjects with bilaterally absent vestibular function. The solid lines are fits to the data by the model shown in Figure 1. PID parameters for the visual loop in both the fixed and sway-referenced conditions are $K_{pv} = 14$ N-m/deg, $K_{iv} = 6$ N-m/deg-s, $K_{dv} = 6$ N-m-s/deg. Parameters for the somatosensory loop are $K_{ps} = 18$ N-m/deg, $K_{is} = 12$ N-m/deg-s, $K_{ds} = 7$ N-m-s/deg. The time delay parameters are 0.2 s for the visual loop and 0.07 s for the somatosensory loop.

and phase data. The transfer function given by Equation 5 only produces a phase lead required to match the actual gain and phase data from vestibular loss subject if the integral component is present in the PID controller. This can be taken as evidence that integral control action is present in the postural control system.

For the fixed support surface condition, the gains were all less than unity but were increasing with increasing frequency. A phase lead was present at 0.1 Hz and a phase lag at 0.5 Hz. However the magnitude of this lead and lag were smaller in the fixed condition compared to the sway-referenced condition. With the visual loop PID parameters remaining fixed at the values that provided a good fit to the sway-referenced condition data, a reasonable fit to the fixed condition experimental data was found by adjusting the somatosensory loop PID parameters. In this case the somatosensory loop time delay was set to 0.07 s. The fixed condition model curve fit is less satisfying than the sway-referenced condition fit in that a resonance peak at about 1.5 Hz is present. While the fit closely approximates the available gain and phase data, the presence of the peak at 1.5 Hz indicates that the system is not far from instability. We currently have no data to confirm the prediction of this resonance peak.

SUMMARY

A simple feedback control model for the control of upright stance in subjects without vestibular function was developed. The model assumes that somatosensory and visual orientation cues are processed independently and then add linearly to produce the corrective torques necessary to maintain upright stance. Different processing

time delays were assumed for the somatosensory and visual systems, but no attempt was made to align the timing of signals from the two systems.

The model is consistent with experimental data from vestibular loss subjects whose posture was perturbed by low amplitude visual surround motion. The model provides some insight into the central processing of sensory motion information since it suggests that a mathematical integration of body sway position occurs in postural control, even though this integration is not strictly required for the maintenance of upright stance (Johansson et al., 1988). The model also demonstrates the potential for resonance effects in postural control with the resonance properties largely dependent upon the processing of sensory orientation information.

An obvious direct extension of this model is to include vestibular feedback by adding a third feedback loop which generates corrective torque that sums linearly with the somatosensory and visual loop torques. However there is some evidence that vestibular information below a frequency-dependent threshold level does not contribute to postural control (Peterka & Benolken, 1992). Since a threshold nonlinearity cannot be linearized in order to calculate transfer function equations, simulations would be required for comparisons with experimental data.

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STABILIZING RESPONSES DURING PITCH AXIS STIMULATION

Section 4 Vestibular threshold effects in ocular and postural reflexes (Peterka)

4.1 Introduction

Stabilizing reflexes are often studied using stimuli with high frequency and large amplitude characteristics that resemble "natural" stimuli such as walking, running, or jumping (Grossman et al. 1988). However many limited motion activities also require reflex control. A good example is the control of quiet upright stance in humans. The maintenance of a stance position requires active control since an upright body is an inherently unstable mechanical system.

Sensory information for the control of quiet stance is potentially available from visual, somatosensory, and vestibular motion receptors. An important question is whether or not vestibular motion cues are actually used for postural control during quiet stance. This question arises because the vestibular thresholds identified in psychophysical experiments (Benson et al. 1989, Benson and Brown 1992) are larger than the motions that typically occur during quiet stance (Goldberg, 1992). In addition, Mergner and coworkers (Mergner et al. 1991, 1993) have shown the important influence of vestibular threshold effects on head, neck, and body motion perception. The presence of a vestibular threshold could have a large and direct influence on the dynamic response characteristics of postural control during quiet stance.

A second question is whether or not vestibular threshold phenomena are specific to a given reflex system (e.g. postural control), or are a general feature of all vestibular reflexes. If thresholds with similar amplitudes were identifiable in all vestibular reflex systems, then a common source for the threshold phenomena is possible, perhaps with a peripheral vestibular origin. If the threshold phenomenon were present in one reflex system but not another, then the threshold phenomenon would be more likely due to the central processing of vestibular motion information. In this later case one might speculate that the centrally created thresholds served some useful purpose in the reflex systems where they were present.

4.2 Identification of threshold effects in postural control

Vestibular-related threshold properties were observed in posture experiments (Peterka and Benolken, 1992) when comparing the amplitude of visually evoked postural sway between subjects with bilaterally absent vestibular function and normal subjects. Body sway was evoked by sinusoidally rotating a full field visual surround in an anterior-posterior direction about an axis near the ankle joint. This stimulus induced body sway which followed the visual surround motion. Fourier methods were used to calculate the amplitude of body sway (specifically rotation of the body's center-of-gravity about the ankle joint) at the stimulus frequency. Subjects were tested at three frequencies of visual surround motion, 0.1, 0.2, and 0.5 Hz, and six different amplitudes ranging from $\pm 0.2^\circ$ to $\pm 10^\circ$. The accuracy of somatosensory orientation

cues was varied using two test conditions. Subjects either stood on a fixed support surface or a sway-referenced support surface. Sway-referencing was accomplished by continuously rotating the platform (axis through the ankle joint) to match the subject's body sway angle as measured at the level of the subject's hips. Assuming that no knee flexion occurred, sway-referencing maintained an unchanging ankle joint angle throughout the testing and therefore eliminated somatosensory orientation cues from muscle stretch and ankle joint motions that would normally be associated with body sway.

In the three vestibular loss subjects tested, visual surround motion evoked body sway that increased monotonically with increasing stimulus amplitude at each of the test frequencies and on both the fixed and sway-referenced conditions. The vestibular loss subjects consistently fell at the larger test amplitudes. In the six normal subjects tested, a monotonic increase in sway was only seen at the lower test amplitudes. At these lower test amplitudes, the amplitude of evoked sway in normals was close to the amplitude seen in the vestibular loss subjects. However at larger stimulus amplitudes, the visually evoked sway in normals reached a saturation level such that body sway amplitude remained unchanged despite increasing visual stimulus amplitude. The saturation sway amplitude decreased with increasing stimulus frequency and was lower in the fixed support surface conditions as compared to the sway-referenced conditions (Figure 1).

The fact that (1) low amplitude visual stimuli evoked nearly the same amplitude sway in both normal and vestibular loss subjects, and (2) the amplitude of visually evoked sway reached a saturation level in normal subjects but not in vestibular loss subjects suggested that a vestibular-related threshold phenomena existed. That is, if sway remained below a certain amplitude, normal subjects were not making use of vestibular information for postural control. Therefore a low amplitude external stimulus (such as a visual stimulus) caused as large a postural disturbance in normal subjects as it did in vestibular loss subjects. However if the stimulus produced a postural disturbance above a certain threshold amplitude, then normal subjects did make use of vestibular information to attenuate the effects of the disturbance while the effects of the disturbance remained unattenuated in vestibular loss subjects.

The amplitude of saturated sway in normal subjects provided a measure of the threshold amplitudes. The identified thresholds were a phenomena associated with the presence of vestibular function. However since head motion was not directly monitored in our experiments, we do not know if body sway was a good indicator of actual head motion. Therefore the observed threshold amplitudes might have either a direct relation to vestibular function (assuming the head was fixed on the body), or they might have an indirect relationship to vestibular function (assuming the head was not fixed on the body and some combination of vestibular and somatosensory information was required to interpret vestibular motion cues).

The threshold amplitudes (in degrees of sway about the ankle joint) decreased in proportion to increasing frequency (Figure 1). This pattern was consistent with the thresholds being a constant rotational velocity threshold with values of about $1.05^\circ/\text{s}$ ($2.1^\circ/\text{s}$ peak to peak) in the sway-referenced condition and about $0.25^\circ/\text{s}$ in the fixed condition. The velocity thresholds identified under the sway-referenced conditions

were similar to perceptual rotational velocity thresholds identified during rotations about an earth vertical axis in the dark (Benson et al. 1989, Benson and Brown 1992). The reduced thresholds in fixed compared to sway-referenced support surface conditions were possibly comparable to reduced perceptual thresholds associated with test conditions which evoked the oculogyral illusion (Benson and Brown, 1989, Clark and Stewart, 1968), or may be related to the apparent changes in thresholds identified during vestibular and proprioceptive system interactions investigated by Mergner and coworkers (Mergner et al. 1993).

Thresholds associated with postural sway could involve both semicircular canal and otolith receptors. The fact that the posturally derived thresholds were consistent with a constant velocity threshold does not necessarily imply that they were associated with semicircular canal rather than otolith function. Over the limited frequency range of 0.1 to 0.5 Hz, an otolith afferent sensitive to acceleration in an anterior-posterior direction (e.g. one whose polarization vector was directed nasally) would have a response to sinusoidal body sway (assuming a rigid head and body motion) that was in phase with sway position, but whose amplitude change with changing frequency was approximately consistent with a rotational velocity sensor. Therefore over the frequency range tested the observed threshold data was compatible with either an otolith or semicircular canal source.

4.3 Identification of threshold effects in eye movement control

Vestibular-related threshold effects might be present in all vestibular reflex systems. This would be consistent with a common source for the threshold, e.g. a peripheral vestibular origin. Alternatively, threshold properties might be tailored by central processes to accomplish specific functional goals of each different reflex system.

We attempted to identify threshold properties in two different vestibulo-ocular reflex (VOR) systems and to compare these results with the vestibular-related thresholds associated with postural control. The two VOR systems were (1) pitch rotations about an earth horizontal axis located 0.75 m below the head, which evoke vertical eye movements, and (2) roll rotations about an earth horizontal axis passing through the head, which evoke torsional eye movements. Both of these stimuli involve concurrent stimulation of the vertical semicircular canals and otolith organs, with the pitch rotation most resembling the postural control situation since both dynamic and static otolith stimulation occur with head off-axis rotations. The pitch rotations were made in complete darkness while the roll rotations were made while viewing a single on-axis LED in an otherwise dark room. Eye movements were recorded by video oculography with vertical and torsional eye movement measures made off-line using image analysis techniques.

The approach used to identify VOR thresholds was different from the one used to identify the posture-related thresholds. Since it was difficult to record eye movements with sufficient accuracy at very low stimulus levels, supra-threshold stimuli were used. The ability of this technique to detect a threshold depends upon the functional characteristic of the threshold element. Figure 2A shows three simple idealized input-output functions which all possess threshold properties. The type (i) function shows an input-output function that is linear

except for a small jump discontinuity near the origin. The region between the points of discontinuity (the threshold levels) has zero slope, and therefore no change in output will occur if the input stimulus is below the threshold amplitude. The type (ii) function shows a linear input-output relation that is displaced from the origin along the abscissa such that there is no jump discontinuity between the below threshold and the above threshold sections of the function. The type (iii) function is a power function. A power function has threshold-like properties since the slope of the input-output function (i.e. the gain of the function) is zero at the origin and is low in the region around the origin.

The experimenter's practical ability to detect the presence of a threshold depends upon the functional type of the input-output relation. Since physiological and measurement noise is always present, it is often not practical to present stimuli and measure responses that are actually below threshold levels. However the use of below threshold level stimuli is probably necessary to definitively identify a type (i) threshold. In contrast, presence of the type (ii) and (iii) thresholds can be identified using above threshold stimulation. For example, if the gain of the response (peak response amplitude divided by stimulus amplitude) is plotted as a function of the stimulus amplitude, the response gain will be close to zero near the threshold and will increase with increasing stimulus amplitude.

Figure 2B shows VOR gain plotted for the two VOR reflexes as a function of stimulus amplitude. Pitch VOR gains did not change as a function of stimulus amplitude for stimuli as low as $1^\circ/\text{s}$. Therefore if there is a type (ii) nonlinearity in this system, the threshold level was below a value of about $0.1^\circ/\text{s}$ (the detection limit is about one decade below the lowest amplitude stimulus), and there was no evidence for a power function response. It was not possible to rule out the existence of a type (i) threshold with a threshold level up to $1^\circ/\text{s}$. In contrast, the roll VOR gains showed a clear increase in gain with increasing stimulus amplitude. The roll VOR results were consistent with a power function response (type (iii) threshold).

4.4 Conclusions

Vestibular-related threshold effects were identified in the human postural control system. During pitch axis rotations that potentially provided patterns of vestibular stimulation similar to those occurring during anterior-posture postural sway, no corresponding threshold was identified in the VOR. However VOR eye movements evoked by roll rotations showed power function responses which effectively have threshold properties. These overall mixed results suggest that vestibular threshold effects do not have a single origin, but rather might be created centrally to accomplish specific tasks. These tasks might include (1) noise reduction and simplification of motor control by excluding low amplitude vestibular signals from participating in reflexes involving sensory system interactions, (2) bias control to prevent motor action due to the presence of erroneous signals such as a small imbalance of vestibular function between the two ears, and (3) promotion of perceptual stability by preventing illusions of movement due to vestibular imbalances.

Acknowledgement

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Figure Legends

Figure 1. The amplitude of saturated center-of-gravity body sway in six normal subjects as a function of visual stimulus frequency and support surface condition (mean \pm 1 standard error).

Figure 2. (A) Three theoretical nonlinear stimulus-response functions with threshold properties. (B) VOR gain as a function of stimulus amplitude for two stimulus conditions: Head off-axis pitch rotations and head on-axis roll rotations. Stimulus frequency in both conditions was 0.2 Hz.

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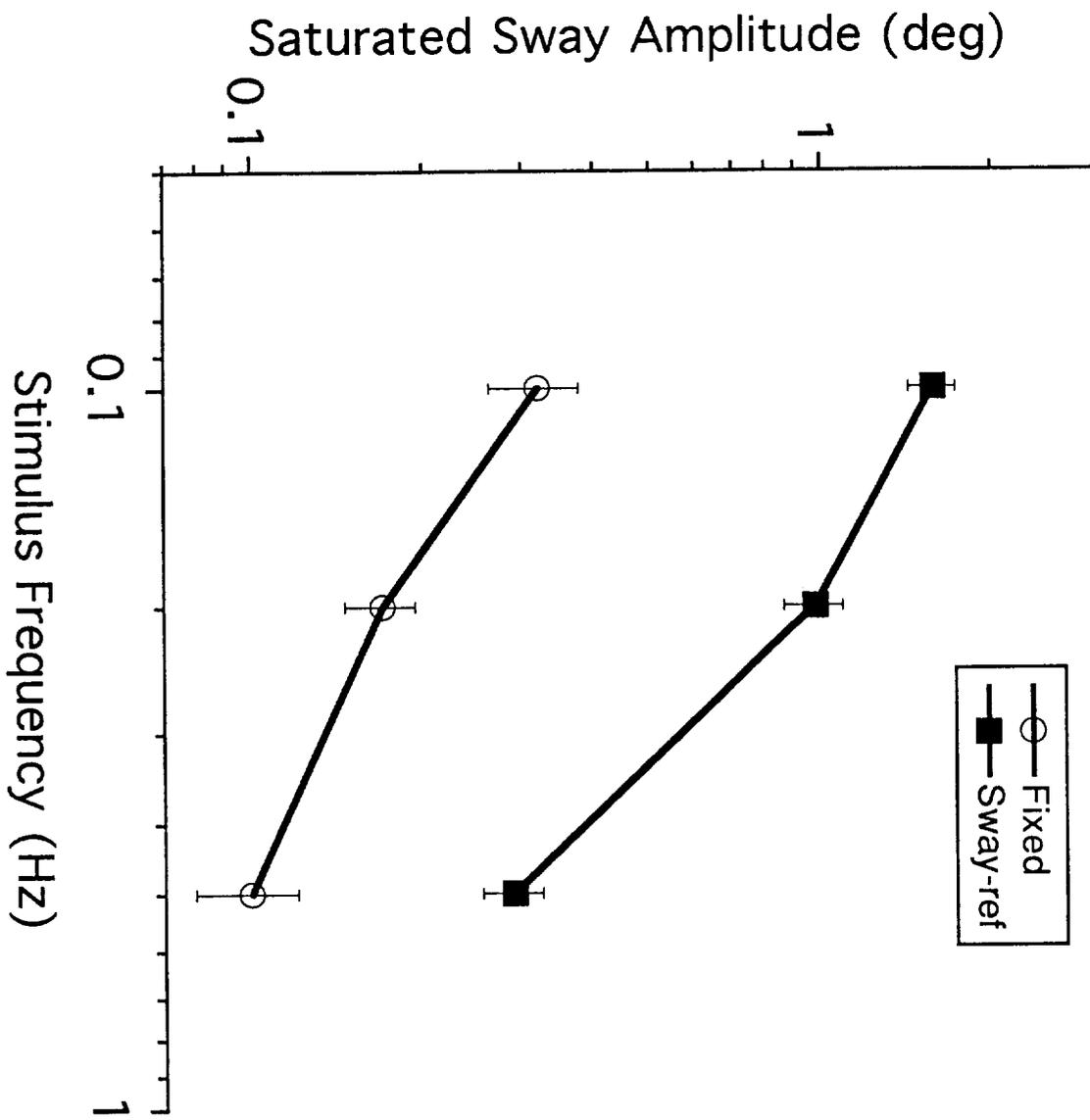


Figure 2.

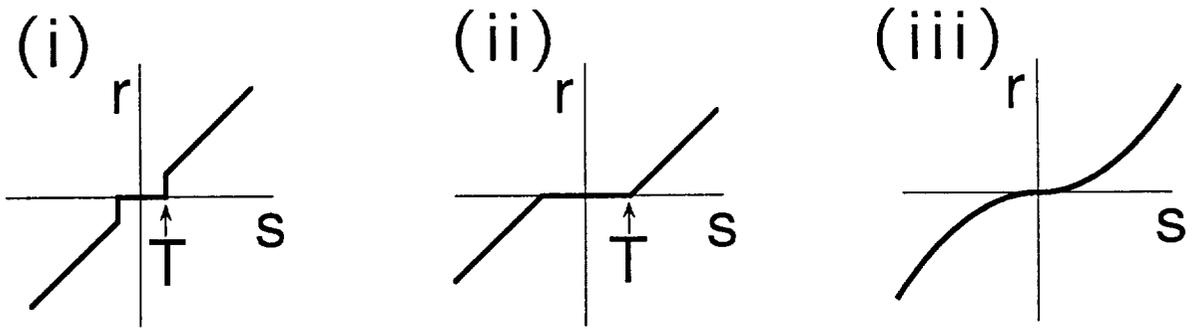
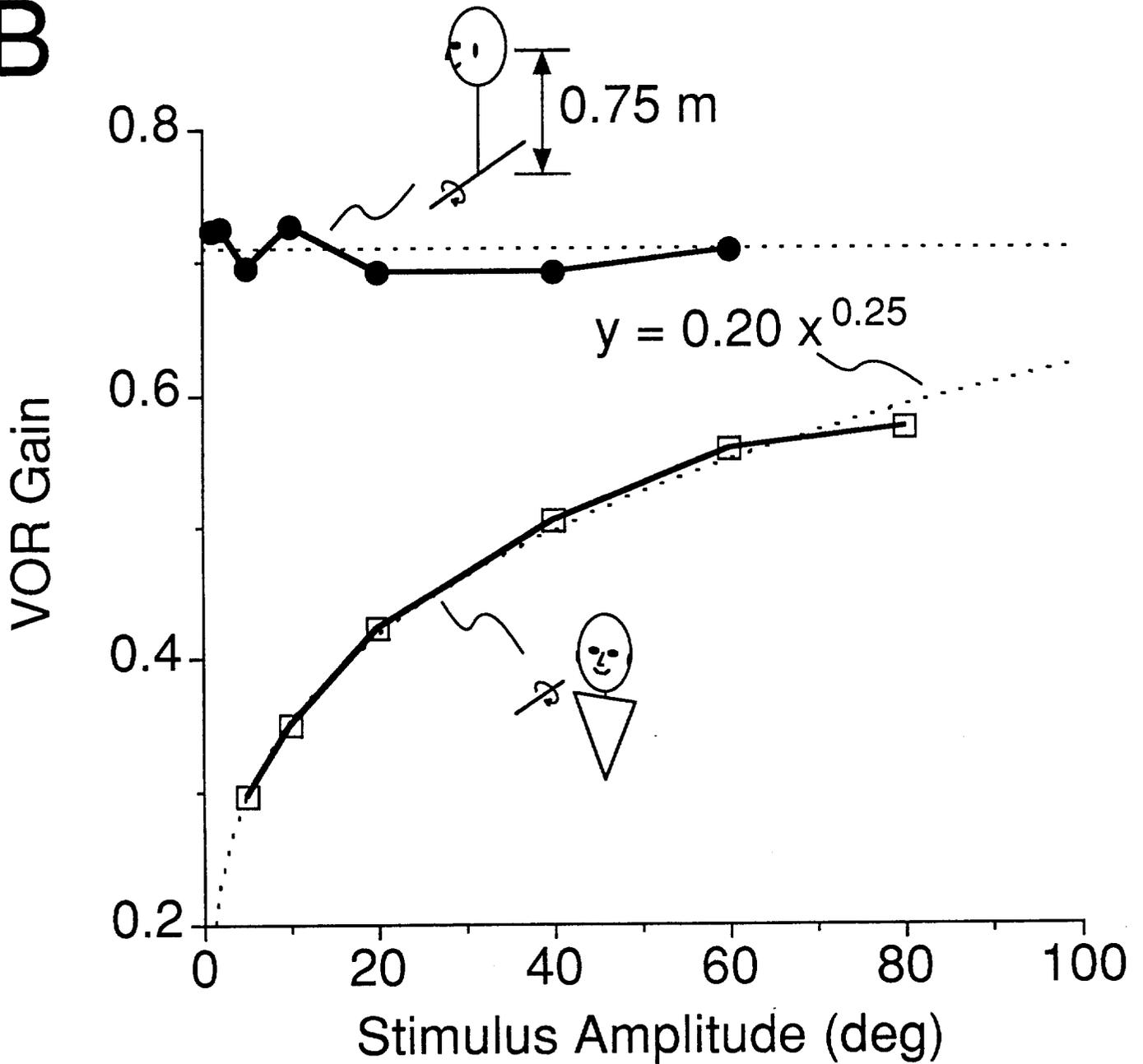
A**B**

Figure 10